

Temporal percolation of the susceptible network in an epidemic spreading

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Abstract

In this work, we study the evolution of the susceptible individuals during the spread of an epidemic modeled by the susceptible-infected-recovered (SIR) process spreading on the top of complex networks. Using an edge-based compartmental approach and percolation tools, we find that a time-dependent quantity $\Phi_S(t)$, namely, the probability that a given neighbor of a node is susceptible at time t , is the control parameter of a node void percolation process involving those nodes on the network not-reached by the disease. We show that there exists a critical time t_c above which the giant susceptible component is destroyed. As a consequence, in order to preserve a macroscopic connected fraction of the network composed by healthy individuals which guarantee its functionality, any mitigation strategy should be implemented before this critical time t_c . Our theoretical results are confirmed by extensive simulations of the SIR process.

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I. INTRODUCTION

The study of epidemic spreading has been one of the most successful applications on networks science. Recent outbreaks of new influenza strains like the H1N1 [1] and the H5N5 flu or the Severe Acute Respiratory Syndrome (SARS) [2], which are characterized by a high rate of mortality and/or fast propagation velocity, motivate the development of epidemic models that capture the main features of the spread of those diseases. In particular, mathematical tools applied to model epidemics are very important since they allow to understand how a disease impact on the society, helping to develop new policies to slow down its spreading.

One of the simplest models that reproduce seasonal diseases, such as influenza, is the susceptible-infected-recovered (SIR) model [3, 4], which has been the subject of extensive theoretical and numerical research on complex networks [3]. In the SIR model the individuals can be in one of three states, susceptible, infected or recovered. In its discrete formulation [5–7], at each time step, infected individuals infect their susceptible neighbors with probability β and recover at a fixed time t_r since they were infected, called recovery time. According to these rules, the disease spreads on the contact network until it reaches the steady state where there are only susceptible and recovered individuals. It was found that the steady state of the SIR model can be mapped into a link percolation problem which provides a theoretical framework to study this process [6, 8–10]. It is known that the size of the infection, defined as the fraction of recovered individuals at the steady state, is governed by the effective probability of infection or transmissibility T of the disease which depends on β and t_r . In the SIR model, the size of the infection is the order parameter of a second order phase transition with a critical threshold transmissibility T_c . Below T_c the disease is an outbreak, where the infection reaches a small fraction of the population while above T_c an epidemic develops exactly as in a link percolation process [6, 8–10]. In uncorrelated infinite networks this threshold is given by $T_c = 1/(\kappa - 1)$ [6, 11], where $\kappa = \langle k^2 \rangle / \langle k \rangle$ is the branching factor of the network, and $\langle k \rangle$ and $\langle k^2 \rangle$ are the first and the second moment, respectively, of the degree distribution $P(k)$. Here, k is the degree or number of links that a node can have with $k_{min} \leq k \leq k_{max}$. For Erdős-Rényi networks (ER), the degree distribution is $P(k) = e^{-\langle k \rangle} \langle k \rangle^k / k!$ and the threshold is found at $T_c = 1/\langle k \rangle$. However, most of the real networks have a heterogeneous degree distribution

that is better represented by a pure Scale-Free network (SF) with $P(k) \sim k^{-\lambda}$, where λ measures the broadness of the distribution. In the thermodynamic limit, for SF networks with $2 < \lambda < 3$, $\langle k^2 \rangle \rightarrow \infty$ and as a consequence, the critical transmissibility $T_c \rightarrow 0$ which means that the epidemic spreads for any value of T [6, 11]. However, due to finite size effects, real networks have finite critical transmissibilities.

In a recent paper, using a generating function formalism, Newman [12] showed that at the steady state of the SIR model there exists a second threshold T^* above which the residual network composed by the biggest giant susceptible cluster that remains after a first propagation, is destroyed. From an epidemiological point of view, this implies that if a disease spreads for a second time on the residual network, it cannot become an epidemic. On the other hand, Valdez *et al.* [13] showed that T^* is an important parameter to determine the efficiency of a mitigation or control strategy, because any strategy that decrease the transmissibility below T^* , can protect a large and connected cluster of susceptible individuals. Using a percolation framework, they explained the lost of the susceptible giant cluster as a not-random node percolation process, that they called node void percolation, in which a susceptible individual corresponds to a void node in link percolation.

Even though percolation theory was very useful to describe the steady state of the SIR model on complex networks, it is still very challenging to explain the dynamics of the model to develop intervention strategies before the epidemic spreads to a large fraction of the population. To describe the dynamics of epidemic spreading on networks, recently some researchers developed differential rate equations for the SIR model that take into account the network topology. Lindquist *et al.* [14] introduced an “effective degree” approach through a large system of ordinary differential equations. Under this approach, the nodes and their neighbors are categorized by their disease state (susceptible, infected, recovered) and each differential equation compute the evolution of the fraction of susceptible or infected nodes with a number i and s of infected and susceptible neighbors, respectively, with $0 \leq i \leq k_{max}$ and $0 \leq s \leq k_{max}$. As a result, a system with $\mathcal{O}(k_{max}^2)$ equations needs to be solved. This approach represents accurately the evolution of the number of infected individuals, but at a high computational cost. On the other hand, Miller [15] and Miller *et al.* [16, 17] proposed an ingenious approach to describe the evolution of a SIR process with rates by means of an edge-based compartmental model

(EBCM) [15, 16] which has the advantage to describe the dynamical spreading of an epidemic with only a few equations. With these equations, the authors found accurate results for the evolution of the number of infected individuals for static and dynamic evolutive topologies like “edge swapping” and “dormant contacts” for transmissibilities above the critical threshold [16].

While most of the literature is focused on studying the evolution of the fraction of infected or susceptible individuals, it has not yet been investigated how the epidemic spread affects the evolution of the network composed by the susceptible individuals. Understanding this problem is important because the network composed by the healthy individuals is the network that sustains the functionality of a society, e.g. the economy of a region. In this paper we present a novel idea for the SIR model, based on a dynamical study of the network composed by susceptible individuals. We show that the temporal decreasing of the size of the giant susceptible cluster can be described as a dynamic void node percolation process with an instantaneous void control parameter. We find that there exists a critical time t_c above which the giant susceptible component overcomes a temporal second order phase transition with mean field exponents. The paper is organized as following: in Methods and Results we present the theoretical framework to derive the evolution equations. Then we study the evolution of the giant susceptible cluster and its temporal critical behavior. Finally we present our conclusions.

II. METHODS AND RESULTS

Theoretical framework

The evolution equations of the dynamic SIR model provide the basis for analyzing theoretically novel magnitudes that could be useful for epidemiologists and authorities to plan policies to stop a disease before an epidemic develops. In the SIR model, initially, all the nodes are susceptible except for one node randomly infected, that represents the index case from which the disease spreads. The infected individual transmits the disease to susceptible neighbors with probability β and recovers t_r time units since he was infected. For the SIR with fixed recovery time, the transmissibility is given by $T(\beta, t_r) \equiv T = 1 - (1 - \beta)^{t_r}$ [13].

In order to study the evolution of the states of the individuals in the SIR with fixed recovery time, we use the edge-based compartmental model (EBCM) [15–17]. The EBCM is based on a generating function formalism, widely implemented in branching and percolation process on complex networks [3, 18–20]. For a branching process that spreads on uncorrelated networks, such as the tree of infected individuals, two generating functions that contain the information of the topology of these networks are defined. The first one is the generating function of the node degree distribution $P(k)$ which is given by $G_0(x) = \sum_k P(k)x^k$. The second one is the generating function of the degree distribution of the first neighbors of a node, also called excess degree distribution $P_1(k) \equiv kP(k)/\langle k \rangle$, given by $G_1(x) = \sum_k kP(k)/\langle k \rangle x^{k-1}$. Here, $P_1(k)$ is the probability to reach a neighbor of a node, following a link. It is straightforward that the mean connectivity of the nodes is $\langle k \rangle = G'_0(1)$.

Denoting the fraction of susceptible, infected and recovered individuals at time t by $S(t)$, $I(t)$ and $R(t)$, respectively, the EBCM approach describes the evolution of the probability that a node (which we call root node) is susceptible. In order to compute this probability, an edge is randomly chosen and a direction is given, in which the node in the target of the arrow is the root, and the base is its neighbor. Disallowing that the root infects the neighbor, $\theta(t) \equiv \theta_t$ is the probability that the neighbor does not transmit the disease to the root, with θ_t given by

$$\theta_t = \Phi_S(t) + \Phi_I(t) + \Phi_R(t), \quad (1)$$

where $\Phi_S(t)$, $\Phi_R(t)$ and $\Phi_I(t)$ are the probabilities that the neighbor is susceptible, recovered, or infected but has not transmitted yet the disease to the root. The probability that a root node with connectivity k is susceptible is therefore θ_t^k and the fraction of susceptible nodes is $S(t) = \sum_k P(k)\theta_t^k = G_0(\theta_t)$. This approach simplifies the calculations, reducing the problem to finding an evolution equation for θ_t , from where the evolution of $S(t)$, $R(t)$ and $I(t)$ is derived. Thus, using the EBCM approach adapted to SIR with fixed t_r (see Appendix Sec.I), the evolutions of θ_t , $\Phi_S(t)$ and $\Phi_I(t)$ are given by the deterministic equations

$$\Delta\theta_t = -\beta\Phi_I(t), \quad (2)$$

$$\Delta\Phi_S(t) = G_1(\theta_{t+1}) - G_1(\theta_t), \quad (3)$$

$$\Delta\Phi_I(t) = -\beta\Phi_I(t) - \Delta\Phi_S(t) + (1 - T)\Delta\Phi_S(t - t_r), \quad (4)$$

where Δ is the discrete change of the variables between times t and $t+1$. Eq. (2) represents the decrease of θ_t when a infected neighbor transmits the disease. Eq. (3) represents the decrease of $\Phi_S(t)$ when a susceptible neighbor is infected (notice that $\Delta\Phi_S(t) < 0$). This term contributes to an increase of $\Phi_I(t)$ in Eq. (4) where the first term represents the decrease of $\Phi_I(t)$ when the links transmit the disease, the second term corresponds to the term of Eq. (3) mentioned above and the third term represents the decrease of $\Phi_I(t)$ due to the recovery of infected individuals.

From the above equations, the evolution of the fraction of infected individuals can be computed as

$$\Delta I(t) = -\Delta S(t) + \Delta S(t - t_r), \quad (5)$$

where the first term represents the fraction of new infected individuals (see Appendix Sec.I). The second term represents the recovery of infected individuals that have been infected t_r time units ago.

These difference equations correctly describe the evolution of $S(t)$, $I(t)$ and $R(t)$ above the criticality for all values of t_r and β (see Appendix Sec.I). In the next section, we will show that combining this approach and dynamic percolation, we can describe the time-dependent evolution of the susceptible individuals in the SIR model as a dynamic void node percolation process for any value of t_r .

A. Temporal percolation of susceptible individuals

In Ref. [13] it was found that the process under which the susceptible clusters size decrease can be explained with node void percolation defined below that as we will show can be related with the dynamic SIR process.

In the steady state of the SIR model an epidemic cluster is equivalent to a Leath growth process [21, 22] with a link occupancy probability T . The Leath process on

complex networks generates a single cluster that represents the infection tree for a given value of the transmission probability T . Denoting by $f_n(T)$ the probability that a cluster reaches the n th generation following a link, the probability $f_\infty(T)$ that a link leads to a giant component ($n \rightarrow \infty$) is given by [13, 22]

$$f_\infty(T) = 1 - \sum_{k=1}^{\infty} \frac{kP(k)}{\langle k \rangle} [1 - T f_\infty(T)]^{k-1}, \quad (6)$$

where $f_\infty(T)$ is the solution of

$$f_\infty(T) = 1 - G_1 [1 - T f_\infty(T)]. \quad (7)$$

As the “infectious” cluster grows from a root, generation by generation, the sizes of the void clusters, *i.e.* the nodes not reached by the disease, are reduced as in a node dilution process, since when a link is traversed a void cluster loses a node and all its edges. As a consequence, for large generations $f_\infty(T)$ can also be interpreted as the probability that a void cluster loses a node. However, in this kind of percolation process the void nodes are not killed at random, instead they are removed following a link. We call this type of percolation “node void percolation”. If we denote by $1 - V^s$ the probability that a void node is removed due to the occupancy of a link, at the steady state the following relation holds

$$1 - V^s = f_\infty(T). \quad (8)$$

Then V^s is the probability that a void node is not removed due to the fact that the link has not been traversed. Thus, V^s is equivalent to $\Phi_S(t \rightarrow \infty)$ because the void nodes correspond to the susceptible individuals in the steady state. As in any percolation process, there is a critical probability V_c^s at which the void network undergoes a second order phase transition. Above V_c^s a giant void component exist while at and below V_c^s void nodes belong only to finite components. In epidemic terms, this means that at V_c^s only finite susceptible clusters can be reached. As a consequence, the fraction of links T^* needed to reach this point fulfills [13]

$$V_c^s = 1 - f_\infty(T^*). \quad (9)$$

Therefore, from Eqs. (7) and (9) we obtain

$$V_c^s = G_1 [1 - T^*(1 - V_c^s)], \quad (10)$$

where T^* is the solution of Eq. (10). This result shows that at the steady state, for $T \geq T^*$, we have $V^s < V_c^s$ and therefore the size of the giant susceptible cluster $S_1 \rightarrow 0$ [13]. Even though static percolation is a useful tool to analyze the final size of the giant component of susceptible individuals [12], it is very important to know the evolution of $S_1(t)$, since it can be used as a criteria to begin or to increase an intervention to protect a large fraction of the susceptible population [13]. As we will show below, $S_1(t)$ can be fully related with a node void percolation process at every instant t .

In order to describe the evolution of the size of the giant susceptible cluster, we define ω_t as the probability that a neighbor of a root not connected to the giant susceptible cluster has not yet transmitted the disease to the root at time t . This is possible if the neighbor of the root node is infected but has not yet transmitted the disease, recovered or susceptible but not connected to the giant susceptible cluster, with probabilities $\Phi_I(t)$, $\Phi_R(t)$ and $G_1(\omega_t)$ respectively. Similarly to θ_t (see Eq. (1)), these probabilities satisfy the relation

$$\Phi_R(t) + \Phi_I(t) + G_1(\omega_t) = \omega_t, \quad (11)$$

where $G_1(\omega_t)$ is the generating function of the neighbor of a root not connected to the giant susceptible cluster. From Eq. (1), $\Phi_I(t) + \Phi_R(t) = \theta_t - \Phi_S(t) = \theta_t - G_1(\theta_t)$. Then Eq. (11) can be rewritten as,

$$\omega_t - G_1(\omega_t) = \theta_t - G_1(\theta_t), \quad (12)$$

and the evolution of $S_1(t)$ is given by

$$S_1(t) = G_0(\theta_t) - G_0(\omega_t), \quad (13)$$

where $G_0(\theta_t)$ is the total fraction of susceptible individuals and $G_0(\omega_t)$ is the fraction of individuals belonging to finite susceptible clusters at time t . Notice that the dynamical Eqs. (12) and (13) are a time-dependent versions of the ones derived in Ref. [12] for the steady state ($t \rightarrow \infty$) of the SIR model. This suggests that the evolution of the giant susceptible or percolating void cluster can be thought as a temporal percolation process. Thus, the magnitudes derived for the static percolation of the susceptible individuals have a dynamical counterpart. As a result, V^s and $\Phi_S(t)$, are equivalent not only at the steady state, but also at every instant of time. In order to show the equivalence, in Fig. 1 we show

in the same plot $S_1(t)$ as a function of $\Phi_S(t)$, obtained from Eqs. (3)-(2) and (12)-(13), and the steady state $S_1(t \rightarrow \infty)$ as a function of V^s [12] for ER and SF networks with the same $\langle k \rangle$ and N for $T = 0.76 > T^*$.

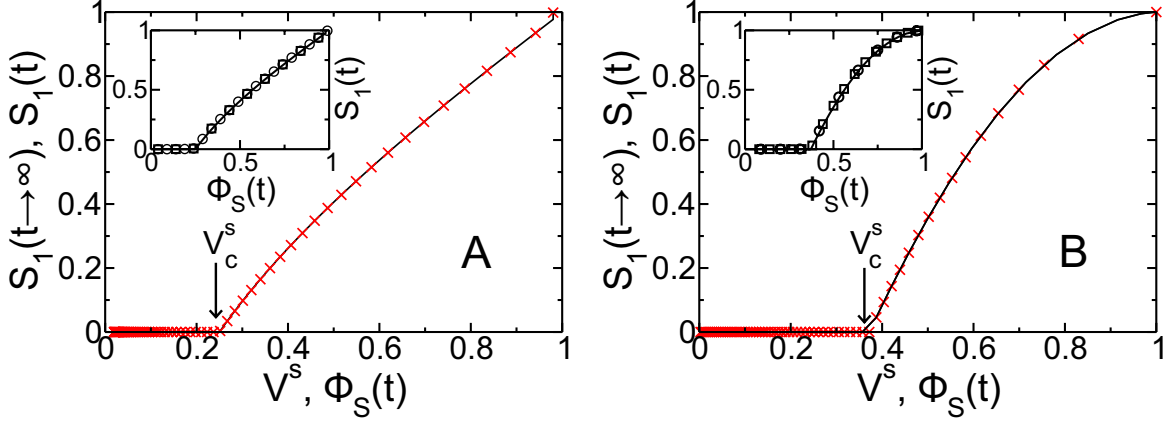


FIG. 1: Equivalence between $\Phi_S(t)$ and V^s . $S_1(t \rightarrow \infty)$ as a function of V^s (\times) obtained in Refs. [12, 13] and $S_1(t)$ as a function of $\Phi_S(t)$ (solid line) obtained from Eqs. (3)-(2) and (12)-(13) with $N = 10^5$ and mean connectivity 4.07 in the giant component for (A) a ER network with $\langle k \rangle = 4$ and (B) SF network with $\lambda = 2.63$, $k_{min} = 2$ and $\langle k \rangle = 4.07$. In the insets we show $S_1(t)$ as a function of $\Phi_S(t)$ from the simulations (symbols) and from Eqs. (3)-(2) and (12)-(13) (solid line) for $t_r = 1$ (\square) and $t_r = 20$ (\circ). (Color online).

As we can see, the static curve $S_1(t \rightarrow \infty)$ as a function of V^s is the same as $S_1(t)$ as a function of $\Phi_S(t)$ and they coincide with the simulations for different values of t_r which shows the equivalence between V^s and $\Phi_S(t)$ at every instant of time and not only at the steady state (for details of the simulations see Appendix Sec.I). Thus our process can be explained by a dynamic percolation with an instantaneous void transmissibility $V^s \equiv \Phi_S(t)$.

With our theoretical formulation, we will show that there is a critical time t_c at which the giant susceptible cluster disappears that correspond to the time at which $\Phi_S(t_c) = V_c^s$. In order to prove this, notice that according to Eq. (12), θ_t and ω_t can be thought as two points with the same image of the function $x - G_1(x)$. Solving this equation for the variable ω_t above T^* , two solutions are found since the curve $x - G_1(x)$ is a concave function for

$x > 0$ as can be seen in Fig. 2. One of the solutions is the trivial one, for which $S_1(t) = 0$, that corresponds to the maximum of the function $x - G(x)$ at $\theta_{t_c} = \omega_{t_c} \equiv \omega_c$. Then the giant susceptible cluster is destroyed at the point ω_c which fulfills

$$[x - G_1(x)]' \big|_{w_c} = 0, \quad (14)$$

then,

$$w_c = \left(G'_1\right)^{-1}(1). \quad (15)$$

Thus when Eq. (14) is satisfied, the giant susceptible cluster disappears and $\Phi_S(t_c) \equiv V_c^s = G_1(\omega_c = \theta_c) = G_1\left[\left(G'_1\right)^{-1}(1)\right]$, *i.e.*

$$\Phi_S(t_c) = G_1\left[\left(G'_1\right)^{-1}(1)\right]. \quad (16)$$

For ER networks it is straightforward to show that $\Phi_S(t_c) = 1/\langle k \rangle$.

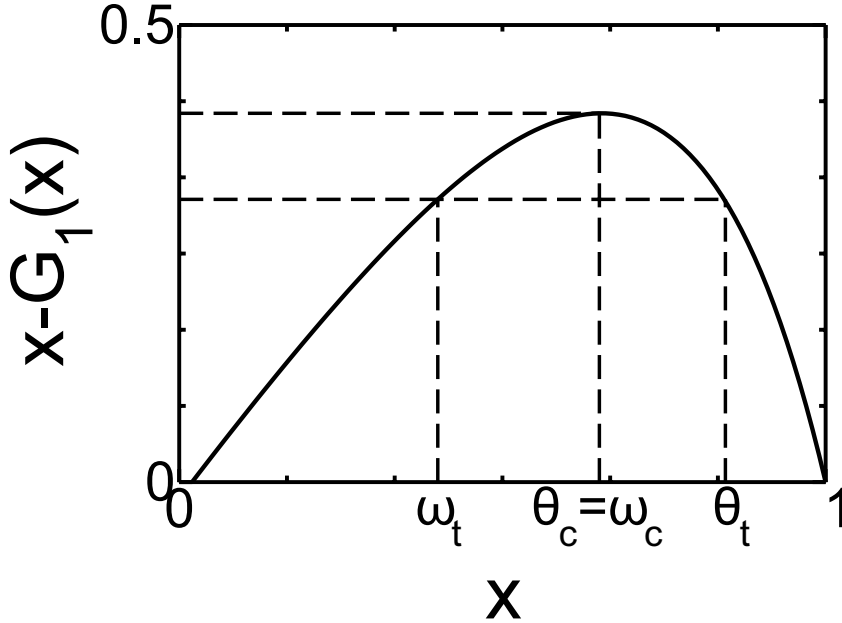


FIG. 2: Schematic of the behavior of Eq. (12) for $T > T^*$. From the initial condition $\theta_0 = \theta(t = 0) = 1$, θ_t and ω_t , satisfies Eq. (12). For $\theta_t \neq \omega_t$ we have two solutions that correspond to $S_1(t) > 0$. When θ_t reaches the maximum of the function $x - G_1(x)$, $\theta_c = \omega_c$, the giant susceptible component is destroyed. The dashed lines are used as a guide to show the possible solutions of Eq. (12).

In Fig. 3 we plot the time evolution of the fraction of susceptible individuals $S_1(t)$ in the susceptible giant component as a function of t for ER and SF networks obtained from the theory and the simulations, for a transmissibility T above T^* .

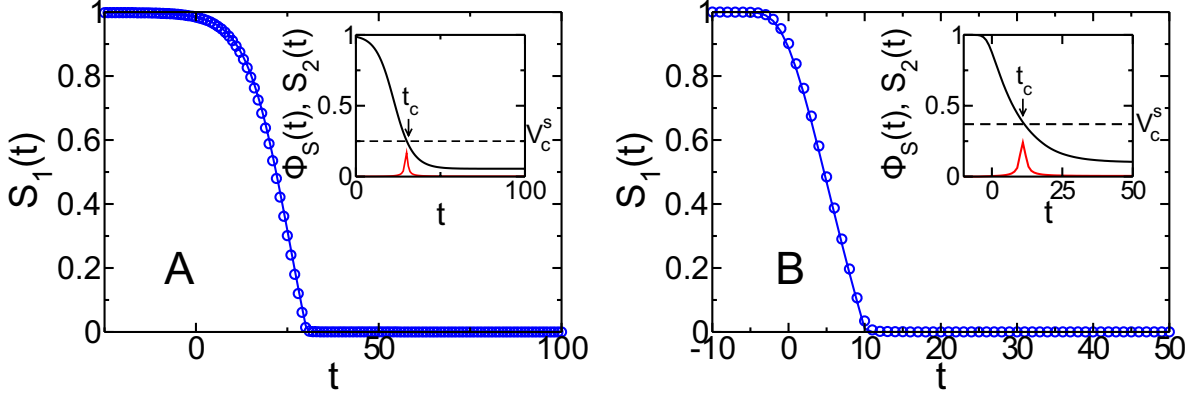


FIG. 3: Time evolution of $S_1(t)$ for $t_r = 20$ and $\beta = 0.07$ ($T = 0.76$) and mean connectivity 4.07 in the giant component for (A) a ER network with $\langle k \rangle = 4$ ($T^* = 0.46$) and (B) a SF networks with $\lambda = 2.63$, minimal connectivity $k_{min} = 2$ and $\langle k \rangle = 4.07$ ($T^* = 0.38$). The symbols correspond to the simulations with the time shifted to $t = 0$ when 1% of the individuals are infected, and the solid lines correspond to the theoretical solutions $S_1(t)$ (blue solid line) of Eqs. (12)-(13). In the insets we show the size of the second biggest susceptible cluster $S_2(t)$ (red solid line) and the evolution of $\Phi_S(t)$ (black solid line) obtained from simulations. The value of $\Phi_S(t_c) = V_c^s$ (dashed line) was obtained from Eq. (16). $S_2(t)$ has been amplified by a factor of 50 in order to show it on the same scale as the rest of the curves. The simulations are averaged over 1000 network realizations with $N = 10^5$. (Color online).

As shown in Fig. 3, there is an excellent agreement between the theoretical curve $S_1(t)$, obtained from Eqs. (12) and (13), and the simulations which validate that percolation tools can be used to describe the time dependence of the susceptible individuals in the SIR process for $T > T^*$. On the other hand, in the figure we can see that for $T > T^*$, the giant susceptible cluster $S_1(t)$ is destroyed at $t = t_c$ which occurs exactly at $\Phi_S(t_c) = V_c^s$

(see the insets of Fig. 3). Our results show that $\Phi_S(t)$ can be used to determine whether a giant susceptible cluster exists at a given time. In turn, in the insets of Fig. 3 we can see that the size of the second susceptible cluster $S_2(t)$ has a sharp peak around t_c , indicating that, as in static percolation, the susceptible individuals overcome a second order phase transition. However, this transition is not given by a random node percolation process. As the disease spreads through the links, the susceptible individuals are removed with probability proportional to $kP(k)$, *i.e.*, the susceptible network loses the higher degree nodes first. For this reason, the disease spreading induces a second order phase transition in the susceptible network with mean field exponents at t_c (see discussion in the Appendix Sec.II).

An important implication of our results is that, it can be used by the health authorities to implement intervention strategies before the critical time t_c is reached. This will allow to protect a macroscopic fraction of the network composed by healthy interconnected individuals which preserve all the topological properties characteristic of social contact networks and their functionality.

Conclusions

In this paper we introduce a temporal dynamic percolation to characterize the evolution of the susceptible individuals in a SIR model. We show using an edge-based compartmental approach and percolation tools that as the disease spreads the evolution of the susceptible network can be explained as a temporal node void percolation that can be mapped instantaneously into static percolation. We show that for transmissibilities above T^* , there exist a critical time above which the giant susceptible cluster is destroyed and the susceptible network overcomes a second order transition with mean field exponents. All our theoretical results are in excellent agreement with the simulations. Our findings are very interesting from an epidemiological point of view since the existence of a threshold time implies that when a very virulent disease reaches a small number of susceptible individuals, the authorities have only a limited time to intervene, in order to protect a big community (susceptible giant component) that has not been already reached by the epidemic, and to preserve the topological features of SF networks. Our finding on the susceptible network could be extended to other epidemics dynamics allowing to obtain a

better description of the effect of diseases spreading on social and technological networks.

III. ACKNOWLEDGMENTS

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Appendix A: The EBCM approach

TABLE S1: Definitions

Variable/Parameter	Definition
β	Infection rate or infection probability.
γ	Recovery rate.
t_r	Recovery time.
T	Transmissibility.
θ_t	Probability that a neighbor of a root node has not transmitted yet the disease to the root node at time t .
$\Phi_S(t)$	Probability that a neighbor of a root node is susceptible at time t .
$\Phi_I(t)$	Probability that an infected neighbor of a root node has not transmitted the disease to the root node at time t .
$\Phi_R(t)$	Probability that a neighbor is recovered at time t without having transmitted the disease to the root node.

In the EBCM approach, θ_t is the probability that a root node has not being infected by a neighbor at time t . This is possible if the neighbor is susceptible, recovered, or infected but has not transmitted the disease yet to the root, which happens with probabilities $\Phi_S(t)$, $\Phi_R(t)$ and $\Phi_I(t)$, respectively. Then, $\theta_t = \Phi_S(t) + \Phi_I(t) + \Phi_R(t)$. The probability that a root node of connectivity k is susceptible is θ^k and the fraction of susceptible

nodes is $S(t) = \sum_k P(k)\theta_t^k = G_0(\theta_t)$. On the other hand, a neighbor is susceptible with probability $\Phi_S(t) = G_1(\theta_t)$. Then in the SIR model with infection and recovery rates [15, 16], the probabilities $\Phi_I(t)$, $\Phi_S(t)$ and θ_t evolve as,

$$\dot{\theta} = -\beta\Phi_I, \quad (\text{S1})$$

$$\dot{\Phi}_S = -\beta G_1'(\theta)\Phi_I, \quad (\text{S2})$$

$$\dot{\Phi}_I = -\beta\Phi_I + \beta G_1'(\theta)\Phi_I - \gamma\Phi_I, \quad (\text{S3})$$

where β and γ are the infection and recovered rates. Eq. (S1) represents the decrease of θ when an infected neighbor transmits the disease. The Eq. (S2) represents the decrease of Φ_S when a susceptible neighbor is infected, which is proportional to $G_1'(\theta)$, *i.e.*, the mean connectivity of the susceptible first neighbors or the excess degree of the susceptible individuals, because when a susceptible individual is infected, all its links except the one used to infected it, can transmit the disease. This term contributes to an increase of Φ_I in Eq. (S3). In Eq. (S3) on the *r.h.s*, the first term represents the decrease of Φ_I when the links transmit the disease, the second term corresponds to the term of Eq. (S2) mentioned above and the third term represents the decrease of Φ_I due to the recovery of infected individuals.

To obtain the evolution of $I(t)$, we use the fact that,

$$\dot{I} + \dot{S} + \dot{R} = 0. \quad (\text{S4})$$

As $\dot{R} = \gamma I$ and $\dot{S} = d(G_0(\theta))/dt = G_0'(\theta)\dot{\theta} = -\beta\Phi_I G_0'(\theta)$, the evolution of the fraction of infected individuals is given by

$$\dot{I} = \beta G_0'(\theta)\Phi_I - \gamma I, \quad (\text{S5})$$

where the first term represents the decrease of S which is proportional to β , the mean connectivity of susceptible individuals $G_0'(\theta)$ and the probability that an outgoing edge from a root is connected with an infected node that has not transmitted the disease to the root at time t . The second term corresponds to the recovery of infected individuals at a rate γ .

We reformulate the EBCM approach process with discrete time steps, for a fixed re-

covery time t_r . It is straightforward that Eq. (S1-S5) can be written as,

$$\Delta\theta_t = -\beta\Phi_I(t), \quad (\text{S6})$$

$$\Delta\Phi_S(t) = G_1(\theta_{t+1}) - G_1(\theta_t), \quad (\text{S7})$$

$$\Delta\Phi_I(t) = -\beta\Phi_I(t) - \Delta\Phi_S(t) + (1 - T)\Delta\Phi_S(t - t_r), \quad (\text{S8})$$

where $1 - T = (1 - \beta)^{t_r}$ denotes the probability that an infected individual has not transmitted the disease to a susceptible individual during t_r time units since he was infected. Finally the evolution of the fraction of infected individuals is given by

$$\Delta I(t) = -\Delta S(t) + \Delta S(t - t_r), \quad (\text{S9})$$

where $-\Delta S(t) = -(G_0(\theta_{t+1}) - G_0(\theta_t))$ represents the fraction of new infected individuals and the second term represents the recovery of infected individuals that have been infected t_r time units ago.

For the simulations we infect only one individual in the giant component of the network and at each time step all the infected individuals infect their susceptible network with probability β and recover at a fixed time t_r since they were infected. We select only the runs in which the size of the epidemic has reached a macroscopic fraction of individuals in the steady state [5, 6, 9] because the deterministic equations are only valid for epidemics above the critical threshold T_c . We performed all the simulations using synchronized or simultaneous updates at each time step.

In Fig. S1, we plot the time evolution of the fraction of infected nodes $I(t)$ for ER and SF networks obtaining by the EBCM approach Eqs. (S6-S9) and the simulation. For the simulations we shifted $t = 0$ to the instant when the disease has reached 1% of the individuals. We choose this reference time, as the time when the disease has reached a size enough to growth deterministically. This choice compensates the time dispersion of each trial around the theoretical solution due to stochastic effects at the early stages of the process when the number of infected nodes is small [16] (see the insets of Fig. S1). As shown in Figs. S1A-B, each trial simulation has the same shape as the theoretical solution which shows that the EBCM approach and the simulations are in excellent agreement.

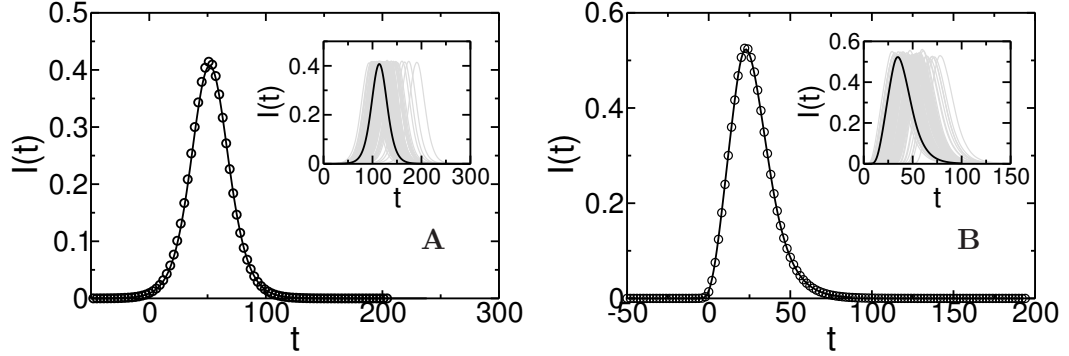


FIG. S1: $I(t)$ for epidemics with $t_r = 20$ and $\beta = 0.04$ ($T = 0.55$) on networks with mean connectivity 4.07 in the giant component, for a ER network with $\langle k \rangle = 4$ (A) and a SF with $\lambda = 2.63$, minimal connectivity $k_{min} = 2$ and $\langle k \rangle = 4.07$ (B). The symbols correspond to an average of one hundred different network realizations with $N = 10^5$ nodes and the solid black curve is the numerical solution of Eq. (S9) shifting the curves to $t = 0$ when 1% of the individuals are infected. The insets show the individual 100 network realizations (solid gray lines) and the numerical solution of Eq. (S9) (solid black line) without the temporal shift transformation.

Appendix B: Node void percolation in the time domain

In node void percolation, as a link is traversed, void node is removed. The void nodes are removed with probability proportional to $kP(k)$. As the susceptible nodes can be mapped into void node percolation, the susceptible network loses their higher degree nodes first as in an intentional attack. As a consequence, the resulting susceptible network is more homogeneous than the original. Thus, mean field exponents of a second order percolating phase transition [23] are expected. In order to show the effect of the disease spreading on the highest degree nodes, in Fig. S2 we plot for a SF network the effective degree distribution of the susceptible nodes obtained from the simulations, in which a susceptible node has degree k when it has k susceptible neighbors.

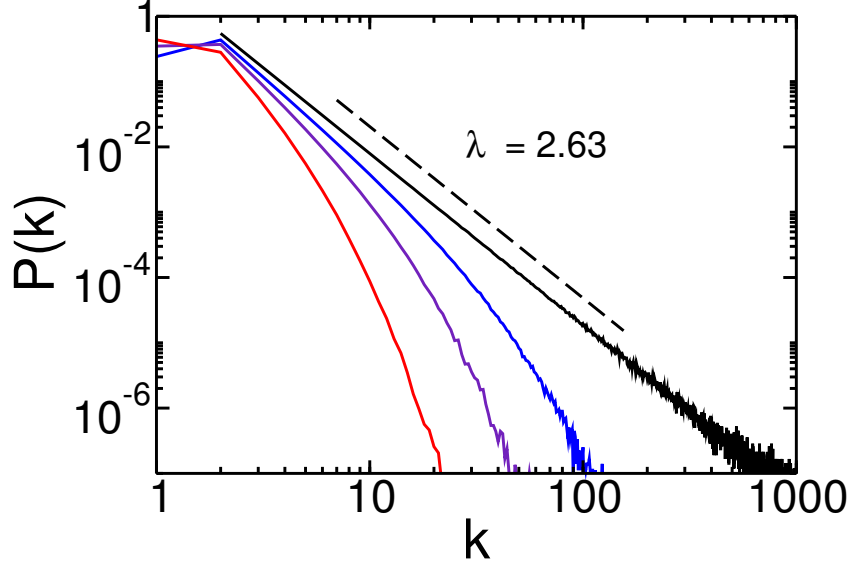


FIG. S2: Simulation results of the degree distribution of susceptible nodes for a SF network with $\lambda = 2.63$, $k_{min} = 2$ and $\langle k \rangle = 4.07$ at different times: at the beginning of the spreading (black solid line), when the disease has reached 10% of individuals (blue solid line), 25% of individuals (violet solid line) and 50% of individuals corresponding to t_c (red solid line). (Color online).

As shown in Fig. S2, as the disease spreads, the effective degree distribution loses the heavy tail. As a result of this process, the susceptible clusters becomes more sparse and at the critical time t_c the topology of the susceptible clusters change drastically since the susceptible individuals lose all the hubs and $P(k)$ has an exponential tail. For percolation in mean field it is known that at the criticality the finite cluster size distribution $n_s \sim s^{-\tau}$ with $\tau = 2.5$ and $S_1[\Phi_s(t)] \sim \Phi_s(t) - \Phi_s(t_c)$. In Fig. S3 we plot the simulations results of the finite size distribution of the susceptible nodes n_s at $t = t_c$.

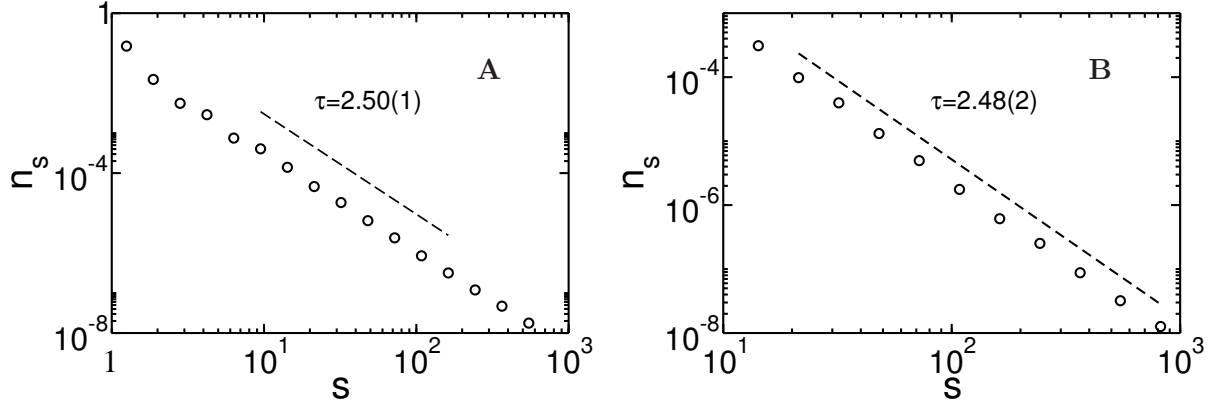


FIG. S3: Log-log of the cluster size distribution n_s of finite susceptible clusters (\circ) at t_c for $t_r = 20$ and $\beta = 0.07$ ($T=0.76$) in a ER network with $\langle k \rangle = 4$ for $t_c = 30$ (A) and a SF with $\lambda = 2.63$, minimal connectivity $k_{min} = 2$ and $\langle k \rangle = 4.07$ for $t_c = 11$. (B). The dashed line corresponds to a power law fitting, from where we obtain an exponent $\tau \approx 2.5$. Our simulations were averaged over 10000 network realizations with $N = 10^5$.

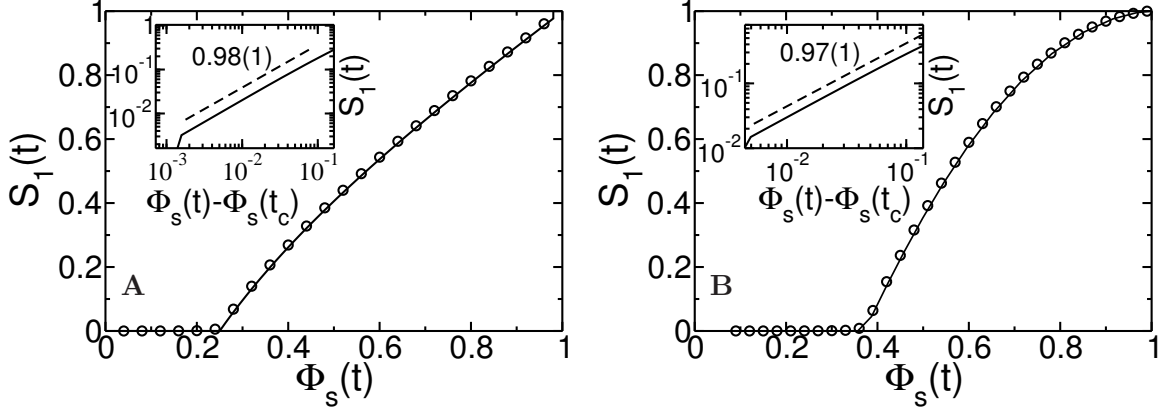


FIG. S4: S_1 as a function of $\Phi_s(t)$ obtained from simulations (\circ) and from the analytical approach (solid line) in a ER network with $\langle k \rangle = 4$ (A) and a SF with $\lambda = 2.63$, minimal connectivity $k_{min} = 2$ and $\langle k \rangle = 4.07$ (B). In the inset we plot S_1 as a function of the distance of $\Phi_s(t)$ to the criticality $\Phi_s(t_c) = V_c$, in log-log scale. The dashed line corresponds to a power law fitting from where we obtain slope ~ 1 . Our simulations were averaged over 1000 network realizations with $N = 10^5$.

We can see that at t_c , $n_s(t_c)$ behaves as a power law with exponent $\tau \approx 2.5$ which corresponds to the mean field value, independently of the initial degree distribution of the network [24]. Similarly, in Fig. S4 we plot $S_1(t)$ as a function of $\Phi_s(t)$ obtained from the simulations and the theoretical approach. We compute $\Phi_s(t)$ from the simulations as the square root of the fraction of edges connecting two susceptible nodes [25]. We can see that $S_1(t)$ behaves as a power law with exponent one with the distance to the criticality $\Phi_s(t_c)$, which also corresponds to the mean field value (see Insets of Fig. S4). Since two critical exponents are sufficient to determine the universality class, the results showed above indicate that in a node void percolation process the susceptible network belongs to the same universality class of mean field percolation and confirms quantitatively the homogenization of the susceptible network during a SIR epidemic spreading.

Finally, in Fig. S5, we plot the critical time t_c , computed from the simulations at $\Phi_s(t) = V_c^s$, as a function of T for different values of t_r . We can see that for the same transmissibility T , when t_r increase, the time to intervene grows since β decrease and

thus the disease spreading is retarded. In turn, when the transmissibility T reaches T^* from above, the critical time t_c grows very fast. This phenomenon is analogous to other second order phase transitions in physics like the relaxation time near the Curie temperature, which are called “critical slowing down” [26, 27], and indicates that once the transmissibility increases slightly above T^* , the time needed to destroy the giant susceptible cluster decreases very fast.

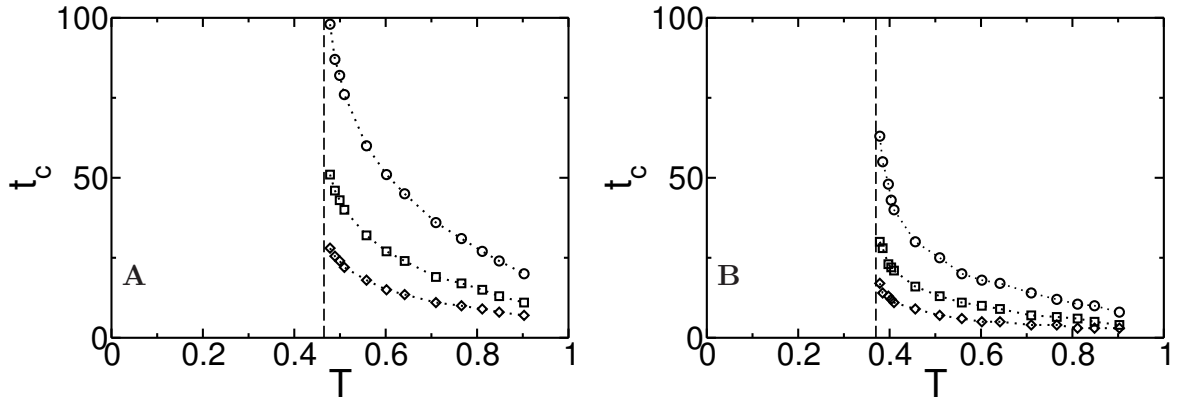


FIG. S5: t_c as a function of T for $\beta = 0.07$ and $t_r = 20$ (\circ), $t_r = 10$ (\square), $t_r = 5$ (\diamond) and mean connectivity 4.07 in the giant component in a ER network with $\langle k \rangle = 4$ ($T^* = 0.46$) (A) and in a SF with $\lambda = 2.63$, minimal connectivity $k_{min} = 2$ and $\langle k \rangle = 4.07$ ($T^* = 0.38$) (B). The dashed line represents the value of T^* . The critical time t_c is measured using $t = 0$ when 1% of individuals are infected. The dotted lines are used as a guide to the eyes.

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- [25] As $\Phi_S(t)$ is the probability that a randomly chosen stub belongs to a susceptible node (conditional on the assumption that stub has not transmitted infection to the node) then the probability that both stubs in a random edge belong to susceptible nodes is $(\Phi_S(t))^2$.
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